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Contents

Foreword

The purpose of the *CDC Surveillance Summaries* is to make available the most current information on conditions of public health interest for which CDC has major responsibility. The reports in this publication complement data provided in the *Morbidity and Mortality Weekly Report (MMWR)* and other CDC publications.

For information on the history of CDC surveillance activities, data sources, and surveillance systems, including the dates of the most recently published reports, refer to *CDC Surveillance Summaries* 1988;37(SS-2), dated June 1988.

Trichinosis Surveillance, United States, 1986

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Trichinella spiralis is a parasite of carnivorous animals that causes the disease trichinosis. In the United States, people become infected by eating poorly cooked pork products or wild animal meat that is infected with the parasite. Although fewer than 100 cases per year are reported to CDC, trichinosis continues to persist as a public health problem in this country. Public health officials believe that the reported cases represent only a fraction of the total number of cases, since many of the mild or asymptomatic infections are undetected or are misdiagnosed unless they are related to more severe cases.

In 1986, 51 cases of trichinosis were reported to CDC from 12 states and the District of Columbia. Thirty-six (71%) of these cases occurred in New Hampshire, Hawaii, Massachusetts, and Pennsylvania. Pennsylvania reported the largest number of cases, 15, or 29% of the total.

In 1986, commercial pork products accounted for only three isolated cases of trichinosis. The other cases of trichinosis caused by pork included wild boar or pork purchased directly from a farm.

Among those cases in which the food item was known or suspected, pork was incriminated in 26 (61%) cases, bear meat in 14 (33%), and other meat in three (7%) cases.

Trichinosis is a preventable disease. The U.S. Department of Agriculture requires that ready-to-eat pork products be heat treated or frozen to kill the parasite before the products are sold to consumers. Fresh pork products, unless they are specifically labeled as "trichinae tested" or "trichinae free," must be cooked to 77°C (170°F), a temperature that will kill the parasite. All wild animal meat must be assumed to be infected with trichinae and also cooked to 77°C (170°F) to safeguard against this disease.

INTRODUCTION

Trichinosis is an infection caused by eating undercooked meat containing larvae of the parasitic worm *Trichinella spiralis*. The larvae are found in various meats, including pork products, wild boar, bear, walrus, and other carnivorous game (*1*). The number of reported cases per year in the United States has declined since CDC first began collecting these statistics in 1947 (*2*) (Figure 1). In the period 1977-1981, the average number of reported cases per year was 137. In 1982-1986, this figure dropped to 57 cases. No apparent change in the surveillance system can account for this downward trend. Although infected pork continues to be the main source of human infection (Figure 2), the decline in the annual incidence of trichinosis over the last 12 years appears to be due to a decrease in cases traceable to commercial pork products

(Figure 3). The absolute number of cases attributed annually to wild animal sources, such as wild boar, bear, and other carnivorous game, has remained relatively constant since 1975 (Figure 2); therefore, although commercial pork is declining as a source of infection, the *percentage* of annual cases due to wild animal meat has increased.

FIGURE 1. Trichinosis cases, by year, United States, 1947-1986

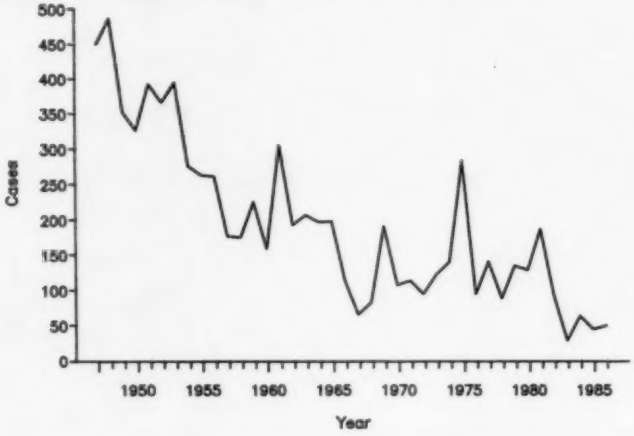
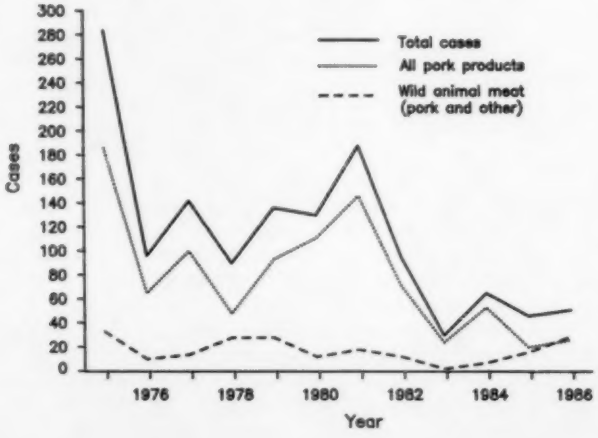


FIGURE 2. Trichinosis cases, by year and source of infection, United States, 1975-1986



MATERIALS AND METHODS

State health departments report new cases of trichinosis by week to the National Morbidity Reporting Service. Supplemental epidemiologic information is submitted by the reporting state on Surveillance Case Report forms (CDC 54.7-Rev 7-81) to the Division of Parasitic Diseases (DPD), Center for Infectious Diseases, CDC. Additional cases are identified through reported results of trichinosis serologic tests performed by the Parasitic Diseases Branch, DPD, and through investigations conducted by the DPD staff.

The CDC case definition for trichinosis is as follows:

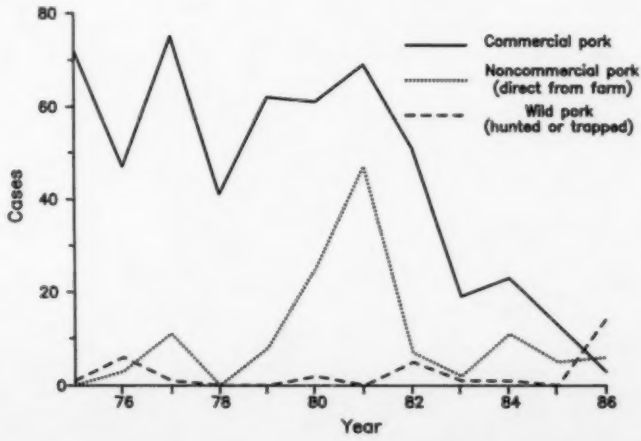
1. *Trichinella*-positive muscle biopsy or positive serologic test for trichinosis in a patient with one or more clinical symptoms compatible with trichinosis, such as eosinophilia, fever, myalgia, and periorbital edema, or
2. In an outbreak, at least one person must meet criterion #1. Associated cases are defined by either a positive serologic test for trichinosis or one or more clinical symptoms compatible with trichinosis (such as eosinophilia, fever, myalgia, and periorbital edema) in persons who have shared the epidemiologically implicated meal or have consumed the implicated meat product.

As in the past, cases reported by the states but that are not accompanied by written surveillance reports or that do not fit the case definition are not included in this report. For data analysis, commercial pork products are defined as pork purchased at supermarkets, butcher shops, or public eating places. Noncommercial pork products are defined as pork purchased directly from a farm. Other pork products are specifically identified according to source, such as hunting or trapping.

RESULTS

In 1986, 51 cases of trichinosis from 12 states and the District of Columbia were reported to CDC. Thirty-six (71%) of these cases occurred in Pennsylvania, New

FIGURE 3. Trichinosis cases, by year and type of implicated pork, United States, 1975-1986



Hampshire, Hawaii, and Massachusetts. Pennsylvania reported the most cases, 15 (29%). The Middle Atlantic and New England states accounted for 62.7% of the reported cases. In 1986, states with the highest trichinosis incidence were New Hampshire (9.0 cases/million population) and Hawaii (6.6). Several states reported 0.1-1.0 case/million population (Table 1).

Of the 51 cases reported in 1986, 64.7% occurred in males and 35.3% in females. The mean age of patients was 36.7 years, ranging from 7 to 81 years (Figure 4).

In previous years, there has been a consistent seasonal pattern for trichinosis in the United States, with a peak in December and January related to the consumption of homemade pork sausage during the Christmas holidays. In 1986, 25 (49%) cases were reported in January and December (Figure 5). Of the 11 cases reported for January, four were traced to a common-source outbreak associated with homemade pork sausage in Massachusetts. The pork used to prepare this sausage came directly from a farm. Five additional cases were traced to a common-source outbreak associated with wild boar in New Hampshire. Of the 14 cases reported for December, 12 were traced to a common-source outbreak associated with bear meat in Pennsylvania.

All but one of the patients reported at least one common sign or symptom of trichinosis: 34 (70.8%) of 48 had fever, 28 (59.6%) of 47 had periorbital edema, 43 (87.8%) of 49 had myalgia, and 31 (91.2%) of 34 had eosinophilia. The patient reporting none of the common symptoms was an 8-year-old boy whose illness was diagnosed by serology during the investigation of an outbreak. One death in 1986 was attributed to trichinosis because myocarditis was found at autopsy.

The mean incubation period for the 35 cases for which the dates of consumption of incriminated meat and the onset of symptoms were available was 19.7 days (range = 1-76 days). A diagnosis of trichinosis was confirmed by serologic tests for 34 persons (81% of those who had serologic tests). Muscle biopsy was performed on nine patients, and seven were positive.

TABLE 1. Trichinosis cases, by state, United States, 1986

State	Cases	Percent	Rate per million population
Arkansas	1	2.0	0.4
California	3	6.0	0.1
District of Columbia	1	2.0	1.6
Florida	1	2.0	0.1
Hawaii	7	3.7	6.6
Iowa	1	2.0	0.3
Massachusetts	5	9.8	0.9
New Hampshire	9	17.6	9.0
New Jersey	3	5.9	0.4
Oregon	1	2.0	0.4
Pennsylvania	15	29.4	1.3
Texas	2	3.9	0.1
Virgina	2	3.9	0.4
TOTAL	**51**	**100**	**0.2**

The suspected meat was examined for *Trichinella* larvae in three outbreaks and in one isolated case. Two of these outbreaks involved wild boar meat (Hawaii, seven cases; New Hampshire, seven cases), and the third involved bear meat (Pennsylvania, 14 cases). The consumption of *Trichinella*-positive cougar meat caused an isolated case in Oregon.

In instances in which the food item was known or suspected (43 cases), pork was incriminated in 26 (60.5%) cases, bear meat in 14 (32.6%), and other meat in three (6.9%). Wild boar was the form of pork most frequently implicated, and it accounted

FIGURE 4. Trichinosis cases, by age group and sex, United States, 1986

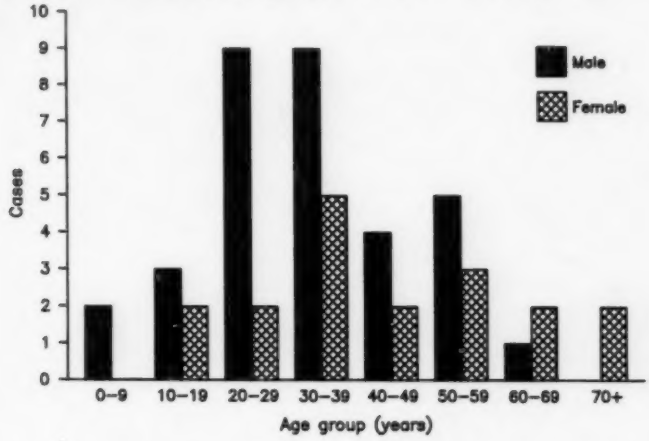
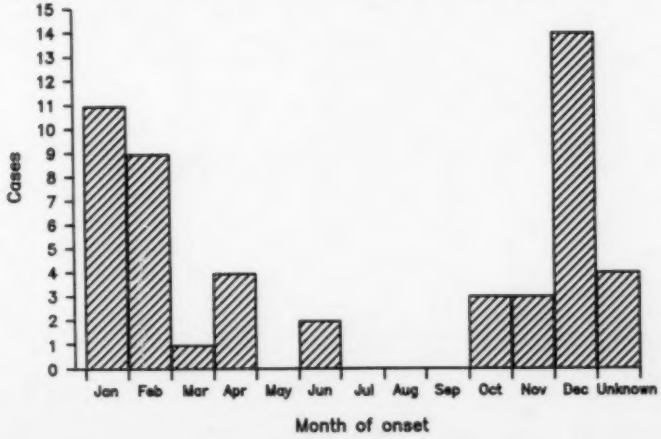


FIGURE 5. Trichinosis cases, by month of onset, United States, 1986



for 14 (32.6%) cases (Table 2). In the other 12 cases due to pork, six patients purchased their pork directly from a farm; three, from a supermarket; and in three cases the origin of the pork was unknown (Table 3).

The method of meat preparation was identified in 42 cases: in 21 (50%), it was roasted over an open fire; in nine (21.4%), the meat was eaten raw; in six (14.3%), it was fried; in five (11.9%), it was microwaved; and in one case, it was boiled.

The source of the meat was identified in 39 cases: in 29 (74.4% of cases for which a source was reported), the implicated meat was wild game; in six (15.4%), it came directly from the farm; and in four (10.2%), the meat came from a supermarket. Of the 29 cases in which the meat was wild game, bear accounted for 14 cases, wild boar for 14, and cougar for one case.

Five common-source outbreaks accounted for 34 (66.7%) of the 51 cases reported to CDC in 1986. Two of the outbreaks involved homemade pork sausage (six cases). In both of these outbreaks, the pork used to prepare the homemade sausage came directly from a farm. The other three outbreaks involved wild animal meat (28 cases). A report on one of these outbreaks, which occurred in Hawaii, has been published (*3*). In that outbreak, signs and symptoms of trichinosis developed in seven of 21 persons who had eaten wild boar meat. The implicated meat was positive for *Trichinella* larvae. Four of the 21 persons ate the meat after it had been microwaved, and the other 17 ate the meat fried. All four who ate microwaved meat became ill; the other three persons had eaten the meat fried.

The largest outbreak, in Pennsylvania, involved 14 persons who ate bear meat at a hunting-season cookout. The meat was served as hamburger or fried steak; examination of remaining meat revealed *Trichinella* larvae. The final outbreak, in

TABLE 2. Trichinosis cases, by source of infection, United States, 1986

Food		Cases	Percent
Pork		26	60.5
Wild Boar	14		
Sausage	7		
Chops	1		
Other	4		
Bear		14	32.6
Cougar		1	2.3
Ground beef		1	2.3
Other		1	2.3
TOTAL		**43**	**100**

TABLE 3. Trichinosis cases due to pork, United States, 1986

Meat Source	Cases	Percent
Commercial pork products	3	11.5
Direct from farm	6	23.2
Game (wild pork)	14	53.8
Unknown/other	3	11.5
TOTAL	**26**	**100**

New Hampshire, involved seven persons who ate ground sausage made from wild boar meat. The sausage was roasted over an open fire at an ice-fishing party.

DISCUSSION

The continuing decrease in the annual incidence of trichinosis in the United States is related to the decline of commercial pork products as a cause of trichinosis (Figure 3). In 1986, commercial pork products were implicated in only three isolated cases of trichinosis.

The exact causes of this decrease are unclear. Since 1980, however, several activities at the national and state level have improved prospects for the control and prevention of trichinosis in commercial pork products. The Federal Swine Health Protection Act (Public Law 96-468) of October 17, 1980, prohibits the feeding of garbage to swine unless the garbage is heat treated to kill disease-causing organisms, including *T. spiralis*. Enforcement of this act is delegated to the individual states contingent on their having adequate laws and regulations and a program for enforcement (*4*). Otherwise, enforcement of the act is the responsibility of the U.S. Department of Agriculture's (USDA's) Animal and Plant Health Inspection Service (APHIS), as is the case in 15 states (unpublished data, D. Galbreath, APHIS). Since 1982, the National Pork Producers Council (NPPC), in cooperation with USDA, has promoted research to develop rapid enzyme-linked immunosorbent assay (ELISA) tests to detect hogs with trichinosis before slaughter, and these tests are being evaluated in the field. Both NPPC and USDA actively promote hog-management practices that avoid factors epidemiologically linked to the transmission of trichinosis; such practices include the immediate removal of dead hogs from feed lots, effective rat control, and other methods that eliminate the interaction of commercial hogs and wild animals.

Some states have implemented programs to eliminate trichinae from their hog populations. An example is Illinois, where feeding garbage to swine is prohibited by state law and where there is an active enforcement program. In addition, the 1986 Illinois Trichinosis Control Act allows the state's Department of Agriculture to quarantine infected herds. It also requires the testing of herds thought to be infected and the depopulation of trichinae-infected herds. Education about proper hog-management practices is also an integral part of the program. Since the passage of this act, the Illinois Department of Agriculture has eliminated trichinae from all but two foci in the state.

The best estimate of the overall prevalence of trichinosis in marketed hogs in the United States is 0.1%. This estimate is based on a study of farm-raised butcher hogs for 1966-1970 (*5*). Recent studies have shown marked regional differences. In a slaughterhouse-based survey of 1,223 hogs in Louisiana, only one (<0.1%) infected animal was detected (*6*). No infection was found among 3,245 hogs from slaughterhouses in Minnesota, Wisconsin, Iowa, South Dakota, and North Dakota examined from 1983 to 1985. The animals examined were from small family farms where pigs were raised for home consumption and from large commercial operations (*7*).

In comparison, 0.7% of 5,315 slaughter hogs from the New England states were infected. Infected hogs were found in five of six states (*8*). In a similar study, 33,482 slaughter hogs from the Mid-Atlantic states were examined. In those states, the sources of hogs varied from small slaughterhouses (fewer than 50 hogs per day) to large commercial operations (more than 4,000 hogs per day). The overall prevalence

was 0.6%, and all the infected hogs were found in the smaller slaughterhouses (fewer than 1,000 hogs per day). In this study, most of the positive samples were obtained from one slaughterhouse, and analysis of the geographic origin of the hogs indicated that most of the infected hogs were from New Jersey and Pennsylvania. Infected hogs could not be traced back to the farm of origin, because there was no identification system for marketed hogs (*9*).

The total elimination of trichinosis from the commercial pork supply ultimately depends on the efforts of individual states to 1) establish active programs to enforce the Swine Health Protection Act, 2) educate hog producers on proper hog-management practices and see that these practices are enforced, and 3) provide mechanisms at the state level to identify and eliminate trichinosis from infected herds. The role of public education concerning the proper cooking of pork to eliminate the risk of trichinosis cannot be overstated. Whereas commercial pork products are declining as a cause of trichinosis, noncommercial sources of pork—such as small farms not using modern hog-management practices—and wild animal meats are emerging as important sources of human trichinosis in the United States. Persons who buy pork from small farms or who eat wild animal meats should be educated as to how to eliminate their risk of acquiring trichinosis.

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Ectopic Pregnancy Surveillance, United States, 1970-1985

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Ectopic pregnancy is now one of the leading causes of maternal death in the United States. In 1984 and 1985, both the numbers and rates of ectopic pregnancy increased. Since the rate of ectopic pregnancy remained unchanged for white women, the rate increase appears to be driven by the increasing rate among women of black and other races. Although ectopic pregnancies accounted for only 1.5% of the total pregnancies in 1984 and 1985, they accounted for 14% of the total maternal deaths in 1984 and for 11% of those deaths in 1985. However, the case-fatality rate for 1985 decreased to 4.2/10,000 ectopic pregnancies, down from the 35.5 deaths/10,000 ectopic pregnancies reported in 1970. Several factors may contribute to the increase in ectopic pregnancies, including heightened awareness of the condition, improved diagnostic technology, and possibly the higher prevalence of risk factors (e.g., acute and chronic salpingitis and sexually transmitted diseases) and the lower prevalence of protective factors (e.g., decreased use of oral contraceptives). Heightened awareness of the condition and improved technology may also be factors resulting in the decreased case-fatality rate.

INTRODUCTION

Ectopic pregnancy is one of the leading causes of maternal deaths in the United States, and it continues to be an important health problem (*1*). This complication of pregnancy results when the fertilized ovum implants anywhere other than the endometrial lining of the uterus (*2*). Ectopic pregnancies represented 1.5% of the total pregnancies in both 1984 and 1985 (*1*). The condition takes its toll in fetal loss, in the number of work days lost during hospitalization and recuperation of young women who are primarily healthy, and in the financial burden of caring for these women.

CDC has previously reported data on ectopic pregnancy for 1970-1983 (*3-5*). This surveillance report includes data for 1984 and 1985.

METHODS

The numbers of ectopic pregnancies presented in this report are estimated from data collected by the National Center for Health Statistics (NCHS) as part of the ongoing National Hospital Discharge Survey (NHDS). The NHDS, which is conducted each year, uses a sample of approximately 400 nonfederal, short-stay hospitals that

represent all 50 states and the District of Columbia. Demographic data, final diagnoses, and surgical procedures shown on the medical record face sheets are abstracted from a sample of medical records from each designated hospital. The more than 200,000 medical records included in the sample every year are weighted to represent approximately 30 million hospital admissions.

The diagnosis of ectopic pregnancy is based on those hospital discharge records with the diagnosis code 631 according to the *International Classification of Diseases, Eighth Revision, Adapted for Use in the United States* (ICDA-8) for 1970-1978 (*6*), and on those abstracts with the diagnosis code 633 according to the *Ninth Revision* (ICD-9) for 1979-1985 (*7*). The number of deaths caused by ectopic pregnancy is based on U.S. vital statistics collected by NCHS. Rates for ectopic pregnancy were calculated by dividing the estimated number of ectopic pregnancies by the total number of reported pregnancies. "Total pregnancies" is defined as the sum of live births, legally induced abortions, and ectopic pregnancies. Data for live births were obtained from NCHS natality statistics (*8*), and data for induced abortions were obtained from CDC's abortion surveillance system. Death-to-case rates were calculated by dividing the number of deaths caused by ectopic pregnancy by the estimated number of ectopic pregnancies. These rates were then reported as deaths per 10,000 cases (*5*). Total "person-days hospitalized" was calculated by multiplying the total estimated number of ectopic pregnancies by the average length of stay for each year.

The U.S. Department of Commerce, Bureau of Census, has defined the four geographic regions of the United States used in this report (Northeast, Midwest, South, West). For the calculation of ectopic pregnancy rates, women were grouped into three age categories: 15-24, 25-34, and 35-44 years of age. For the analysis of mortality from ectopic pregnancy, women were grouped into six age categories: 15-19, 20-24, 25-29, 30-34, 35-39, and 40-44 years. Race-specific rates for the categories "white" and "black and other" were used. If race was not included in the medical records, ectopic pregnancies were redistributed according to the racial distribution of cases for which race was recorded. Estimates of the number of ectopic pregnancies have been rounded to the nearest hundred. The rounding and redistribution of cases with unknown race sometimes cause the sum of numbers to be different from the total. Rates were calculated from the unrounded estimates.

RESULTS

In 1984 and 1985, the numbers and rates of ectopic pregnancies increased, continuing the previously reported trend (Table 1, Figure 1). Sixty-one percent of the ectopic pregnancies in 1984 and 1985 occurred among 25- to 34-year-old women. In 1984 and 1985, the rates of ectopic pregnancies for white women remained unchanged (13.6/1,000 reported pregnancies in 1983, 13.5 in 1984, and 13.3 in 1985). However, the rates for women of black and other races increased from 15.5/1,000 in 1983 to 19.3 in 1984 and to 21.3 in 1985. In 1983, women of black and other races had a risk of ectopic pregnancy 1.1 times higher than that of white women. The risk among women of black and other races rose to 1.4 times that among white women in 1984 and to 1.6 times that among white women in 1985.

For the period 1970-1985, approximately 716,800 ectopic pregnancies occurred among women ages 15-44 in the United States; the overall rate was 10.0/1,000 reported pregnancies. From 1970 through 1985, the total number of ectopic pregnancies increased more than fourfold, from an estimated 17,800 in 1970 to 78,400 in 1985.

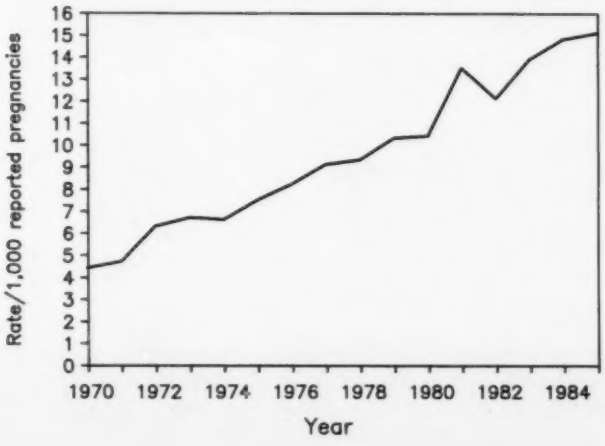
The rate for all women combined increased more than threefold, from 4.5 in 1970 to 15.2 in 1985. When stratified by race, the rates increased more than threefold both for white women (from 4.0 in 1970 to 13.3 in 1985) and for women of black and other

TABLE 1. Numbers and rates of ectopic pregnancies, by year, United States, 1970-1985

Year	Number*	Rate†
1970	17,800	4.5
1971	19,300	4.8
1972	24,500	6.3
1973	25,600	6.8
1974	26,400	6.7
1975	30,500	7.6
1976	34,600	8.3
1977	40,700	9.2
1978	42,400	9.4
1979	49,900	10.4
1980	52,200	10.5
1981	68,000	13.6
1982	61,800	12.3
1983	69,600	14.0
1984	75,400	14.9
1985	78,400	15.2

*Rounded to nearest hundred.
†Rate per 1,000 reported pregnancies (live births, legally induced abortions, and ectopic pregnancies).

FIGURE 1. Ectopic pregnancy rates, by year, United States, 1970-1985



races (from 7.1 in 1970 to 21.3 in 1985). When numbers of ectopic pregnancies were combined into 4-year periods (1970-1973, 1974-1977, 1978-1981, and 1982-1985) and stratified by race, the rates for each racial group had increased from the earliest years (1970-1973) to the latest years (1982-1985) by the following factors: between twofold and threefold for all races combined, almost threefold for white women, and almost twofold for women of black and other races (Table 2). As reported earlier (*5*), the risk of ectopic pregnancy increased with age and was highest for women 35-44 years old (Table 3, Figure 2). White women 35-44 years of age had a threefold higher risk of

TABLE 2. Numbers and rates of ectopic pregnancies by race* and 4-year periods, United States, 1970-1985

Year	White	Rate†	Black and other	Rate	All races	Rate
1970-1973	61,400	4.8	25,600	8.8	87,000	5.6
1974-1977	95,300	7.4	37,000	10.2	132,200	8.0
1978-1981	150,200	10.1	62,100	14.3	212,400	11.0
1982-1985	206,200	13.3	79,000	16.7	285,200	14.1
1970-1985	**513,100**	**9.2**	**203,700**	**13.0**	**716,800**	**10.0**

*Race "unknown" redistributed according to the percentage of race known, and numbers rounded to the nearest hundred. This redistribution and rounding sometimes cause the sum of individual cells not to equal the total.
†Rate per 1,000 reported pregnancies (live births, legally induced abortions, and ectopic pregnancies).

TABLE 3. Numbers and rates of ectopic pregnancies, by race* and age group, United States, 1970-1985

Race	Age group (years)	Number†	Rate§
White	15-44	513,100	9.2
	15-24	161,900	5.6
	25-34	298,400	12.3
	35-44	52,800	17.0
Black and other	15-44	203,700	13.0
	15-24	66,100	7.1
	25-34	111,200	21.0
	35-44	26,500	28.2
All races	15-44	716,800	10.0
	15-24	228,000	6.0
	25-34	409,600	13.9
	35-44	79,200	19.5

*Race "unknown" redistributed according to the percentage of race known. This redistribution and rounding sometimes cause the sum of individual cells not to equal the total.
†Rounded to the nearest hundred.
§Rate per 1,000 reported pregnancies (live births, legally induced abortions, and ectopic pregnancies).

ectopic pregnancy than white women 15-24 years of age, whereas women of black and other races 35-44 years of age had a fourfold higher risk than women of black and other races ages 15-24.

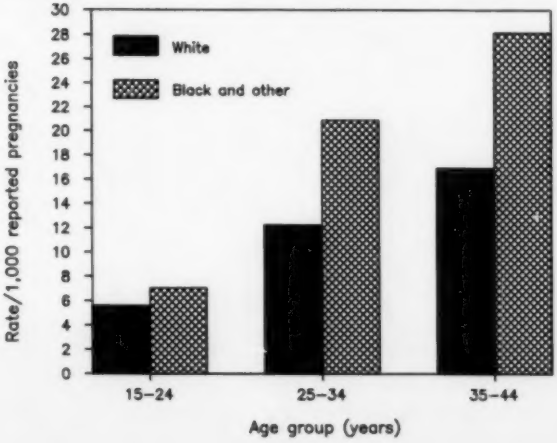
Overall, during the period 1970-1985, the rates of ectopic pregnancies for the four geographic regions were similar; the highest rates occurred in the Midwest and West (Table 4). However, race- and region-specific rates varied. For white women, the rate was highest in the West; for women of black and other races, the rate was highest in the Midwest.

In 1983, the average reported length of hospital stay for women who had ectopic pregnancies was 4.8 days. In both 1984 and 1985, the average length of stay was 4.5 days, a decrease from previous years. In 1984, ectopic pregnancy accounted for 339,300 total person-days of hospitalization, and in 1985 that figure increased to 352,800. Both figures represent increases over previous years and reflect the continued rise in the reported numbers of ectopic pregnancies. The average length of stay for the period 1970-1985 was 5.6 days, and the total person-days of hospitalization during that period was 4,019,100.

In 1984, 39 maternal deaths (14% of all such deaths) were related to ectopic pregnancy (Table 5). In 1985, this figure dropped to 33 (11%). The death-to-case rate decreased to 5.2 in 1984 and to 4.2 in 1985, down from the 5.3 reported in 1983 (Figure 3). In 1984 and 1985, women of black and other races had a death-to-case rate that was four times higher than that for white women.

Between 1970 and 1985, a total of 716 women died as a result of an ectopic pregnancy. Overall, the death-to-case rate has decreased between eightfold and ninefold since the first reporting period in 1970. Women of black and other races continue to have a higher rate of death related to ectopic pregnancy. For the 16-year reporting period, the case-fatality rate for women of black and other races was 3.5 times higher than that for white women (Table 6). Teenagers of black and other races

FIGURE 2. Ectopic pregnancy rates, by race and age group, United States, 1970-1985



had the highest rate of death related to ectopic pregnancy. The rate for this group was 5.5 times higher than that reported for white teenagers (Figure 4).

DISCUSSION

Overall, the number and rate of ectopic pregnancies have increased every year from 1970 through 1985. Chow et al. have noted that the increased incidence may be the result of higher prevalence of risk factors for this condition, lower prevalence of protective factors, or both (*9*). These factors are discussed below.

Recent trends have enabled health-care providers to become more aware of ectopic pregnancy and of the symptoms and signs that may occur among women of childbearing age. Additionally, technologic advances (such as ready availability of quantitative serum pregnancy hormone tests, improved imaging techniques [ultrasound], and laparoscopy for diagnosis and treatment) have led to the earlier diagnosis of some ectopic pregnancies and, ultimately, earlier intervention and better outcome. Although heightened awareness and technology may result in earlier diagnosis and management, they alone cannot explain the large increase in the number of ectopic pregnancies. Another possible factor contributing to the increased frequency is that, in recent years, many women have postponed childbearing until the period of life in which the risk of ectopic pregnancy is highest (*10*).

Ectopic pregnancy was first described in the 10th century (*11*). The etiology of the condition has been variously described as involving multiple maternal and embryonic factors (*2,11,12*). The effects that contraceptive methods may have on susceptibility to ectopic pregnancies have been extensively studied. Pregnancies among women who have used oral contraceptives in the past are not more likely to be ectopic than are pregnancies among women who have never used birth control pills (*13*). Studies on the use of pills at the time of conception have suggested an increased risk of

TABLE 4. Numbers and rates of ectopic pregnancies, by race* and geographic region, United States, 1970-1985

Race	Region	Number†	Rate§
White	Northeast	101,300	8.4
	Midwest	135,800	9.2
	South	139,100	8.4
	West	137,000	10.6
Black and other	Northeast	45,300	13.0
	Midwest	45,700	16.0
	South	88,000	13.3
	West	24,700	10.4
All races	Northeast	146,600	9.5
	Midwest	181,500	10.3
	South	227,100	9.8
	West	161,600	10.6

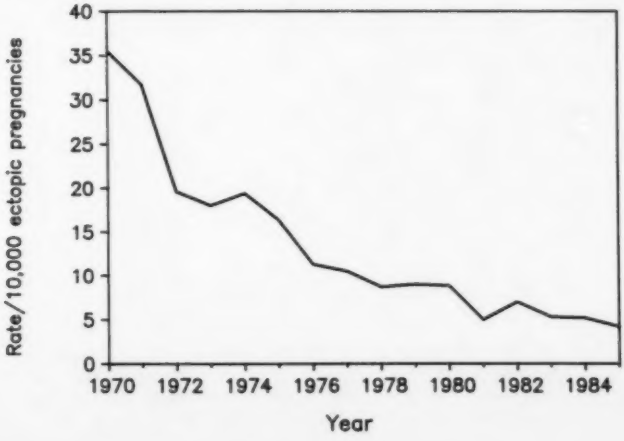
*Race "unknown" redistributed according to the percentage of race known. This redistribution and rounding sometimes cause the sum of individual cells not to equal the total.
†Rounded to the nearest hundred.
§Rate per 1,000 reported pregnancies (live births, legally induced abortions, and ectopic pregnancies).

TABLE 5. Numbers of deaths due to ectopic pregnancy and death-to-case rates, by race and year, United States, 1970-1985

	Number			Rate*		
Year	White	Black and other	Total	White	Black and other	Total
1970	28	35	63	21.7	72.1	35.5
1971	21	40	61	15.1	74.9	31.7
1972	28	20	48	16.2	27.7	19.6
1973	25	21	46	15.1	23.4	18.0
1974	20	31	51	10.1	47.0	19.4
1975	19	31	50	8.8	34.9	16.4
1976	11	28	39	4.4	28.7	11.3
1977	15	29	44	5.2	24.5	10.8
1978	13	24	37	4.4	18.7	8.7
1979	20	25	45	5.7	17.2	9.0
1980	22	24	46	6.0	15.4	8.8
1981	15	19	34	3.1	9.7	5.0
1982	19	24	43	3.8	19.3	7.0
1983	17	20	37	3.3	11.2	5.3
1984	14	25	39	2.7	10.8	5.2
1985	11	22	33	2.1	8.4	4.2
Total	**298**	**418**	**716**	**5.8**	**20.5**	**10.0**

*Deaths from ectopic pregnancy per 10,000 ectopic pregnancies.

FIGURE 3. Ectopic pregnancy mortality rates, by year, United States, 1970-1985



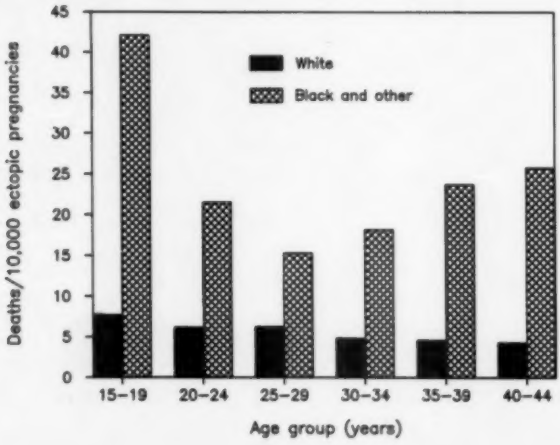
ectopic pregnancy among progestogen-only (minipill) users (*14*). Other studies in which the type of pill was not specified showed either no effect or a protective effect against ectopic pregnancy (*9*).

TABLE 6. Death-to-case rates for women with ectopic pregnancy, by race, age group, and geographic region, United States, 1970-1985

	White		Black and other		Total	
	No.*	Rate†	No.	Rate	No.	Rate
Age group (years)						
15-19	25	7.7	49	42.0	74	16.8
20-24	80	6.2	118	21.7	198	10.8
25-29	113	6.2	102	15.6	215	8.7
30-34	56	4.8	85	18.5	141	8.7
35-39	20	4.6	50	23.8	70	10.9
40-44	4	4.3§	14	25.8§	18	12.2
Region						
Northeast	66	6.5	95	21.0	161	11.0
Midwest	66	4.9	96	21.0	162	8.9
South	88	6.3	187	21.2	275	12.1
West	78	5.7	40	16.2	118	7.3
Total	**298**	**5.8**	**418**	**20.5**	**716**	**10.0**

*Deaths from ectopic pregnancy.
†Deaths from ectopic pregnancy per 10,000 ectopic pregnancies.
§Rates based on <10,000 ectopic pregnancies and thus should be viewed with caution.

FIGURE 4. Ectopic pregnancy death-to-case rates, by race and age group, United States, 1970-1985



Because intrauterine devices (IUDs) decrease the overall incidence of pregnancy, they have been shown to be protective against ectopic pregnancy. Although IUDs are protective against all pregnancies, they have been associated with a higher risk of ectopic pregnancy among women who conceive and do not abort (spontaneously or by induced abortion) while they are still using an IUD (*9*). Laboratory studies suggest that this association may be due either to physiologic changes in tubal motility or ovum transport or to hormonal influences (*9*). One large case-control study showed no difference in the risks of ectopic pregnancy for users and for nonusers of IUDs (*15*). In addition, the risk was lower for persons with a current in situ IUD than for those with a history of IUD use (*15*). This relationship may be explained by the link between IUD use and the occurrence of acute and chronic salpingitis. The prevalence of salpingitis, in relation to ectopic pregnancies, has been shown to vary widely (20%-92%) (*5,9*). In view of this association and of the fact that sexually transmitted diseases (STDs) appear to increase the risk of salpingitis, exposure to the organisms that cause STDs might also increase the risk of ectopic pregnancy.

Barrier contraceptives appear to decrease the risk of ectopic pregnancy by decreasing the occurrence of both pregnancy and STDs (*16*).

With more women choosing tubal sterilization as a means of contraception, scientific knowledge of the long-term risk of ectopic pregnancy with this method will increase. Although poststerilization pregnancies are infrequent, the ones that do occur are more likely to be ectopic than pregnancies among women who are not sterilized. In one study, 176 pregnancies occurred among 37,100 women who had undergone tubal sterilization (*13*). The incidence of ectopic pregnancy for these pregnancies was 16% (n = 28) for all sterilization methods and 20% (n = 14) for those performed by laparoscopy. Overall, these small numbers appear to account for very little of the large increase in the number of ectopic pregnancies.

In 1983, the risk of ectopic pregnancy for women of black and other races was decreasing toward being equal to the risk for white women. By 1985, however, the risk for women of black and other races had increased from 1.1 in 1983 to 1.6 times the risk for white women (*3*). In addition to this widening gap, black and other teenagers continue to have a higher rate of deaths related to ectopic pregnancy, 5.5 times that of white teenagers. Factors including timing and quality of prenatal care are often related to such deaths. Even though new technology may be helping to eliminate many deaths caused by ectopic pregnancies, women of black and other races and younger women, who tend to have less and later prenatal care, are not benefitting from this technology as much as other women (*17,18*).

The number of ectopic pregnancies reported here, although on the increase, may be underreported. The NHDS does not include medical records of patients discharged from federally operated hospitals, such as Armed Forces and Public Health Service hospitals. Furthermore, some ectopic pregnancies are known to resolve spontaneously and, so, are never diagnosed; and with some new treatment methods, hospitalization is not required. On the other hand, total pregnancies used as denominators are underestimated, since spontaneous abortions and stillbirths are not included. Moreover, the number of induced abortions reported to CDC have consistently been less than the numbers reported by private sources who obtained data by direct survey of providers of legal abortion (*19*). As a result of a smaller denominator, the rates of ectopic pregnancy may be elevated. However, because the

method used for collecting data was consistent over the 16-year study period, the data reported here are believed to reflect actual occurrences in the United States.

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Maternal Mortality Surveillance, United States, 1980-1985

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To better define the incidence, causes, and risk factors associated with maternal deaths, the Division of Reproductive Health, Center for Chronic Disease Prevention and Health Promotion, CDC, coordinated a study by the Maternal Mortality Collaborative, a Special Interest Group of the American College of Obstetricians and Gynecologists (ACOG). In 1983, this group established voluntary surveillance of maternal deaths for the years 1980-1985. The Maternal Mortality Collaborative reported 601 maternal deaths from 19 reporting areas for 1980-1985, representing a maternal mortality ratio of 14.1 per 100,000 live births. Overall, 39% more maternal deaths were reported by the Maternal Mortality Collaborative than by the National Center for Health Statistics for these reporting areas. Overall, women over 30 years of age had a higher risk of dying than did younger women. For each age group, women of black and other races had a greater risk of dying than white women, with women of black and other races who were 30 years and older having the highest risk. The leading causes of maternal deaths were embolism, hypertension in pregnancy, sequelae from ectopic pregnancy, hemorrhage, cerebrovascular accidents, and anesthesia complications. Of the 111 nonmaternal deaths, 90 (82%) were attributed to unintentional or intentional injuries. As a result of the success of this voluntary reporting system, the Division of Reproductive Health initiated National Pregnancy Mortality Surveillance in January 1988.

INTRODUCTION

In April 1987, a major effort was proposed to prevent maternal deaths worldwide (*1*). In the United States, the Public Health Service's 1990 health objectives for the nation include lowering maternal mortality for all ethnic groups and geographic areas (*2*). Although maternal mortality is recognized as a public health concern, the magnitude of the problem is often understated, on the basis of routine vital statistics (*3*). The most recently published epidemiologic study of nationwide maternal mortality provided data on deaths that occurred from 1974 through 1978 (*4*).

The Center for Chronic Disease Prevention and Health Promotion (CCDPHP), CDC, coordinated a study conducted by the Maternal Mortality Collaborative, a Special Interest Group of the American College of Obstetricians and Gynecologists. This group consisted of representatives from state maternal mortality committees and

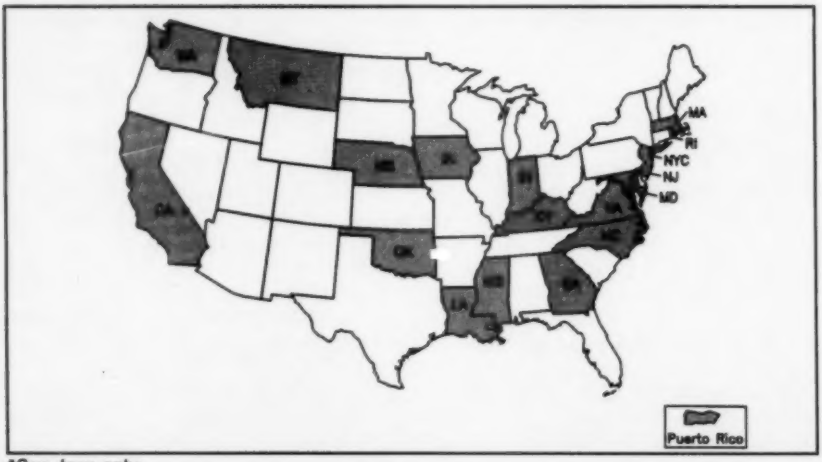
health departments. In 1983, this group established voluntary surveillance of maternal deaths for the years 1980-1985 to improve the detection of maternal deaths, to collect and analyze information, and to disseminate the findings to clinicians. Data were submitted to CDC through the chairperson of the Maternal Mortality Special Interest Group on an abstract form developed for this purpose. A preliminary report on the first 39 maternal deaths from this system was published in 1985 (*5*).

MATERIALS AND METHODS

Between April 1983 and May 1987, reports of maternal deaths for selected years between 1980 and 1985 were contributed voluntarily by collaborators from 19 reporting areas of the United States (16 states; New York City; Puerto Rico; and San Jose, California) (Figure 1). According to The National Center for Health Statistics (NCHS), deaths from these 19 areas represented approximately one-third of the maternal deaths in the United States for 1980-1985 (*6-11*).

After a review of all information provided for each death by the collaborators, a cause of death was assigned to each case according to the *International Classification of Diseases, Clinical Modification, Ninth Revision* (ICD-9). The deaths were then classified as maternal (either direct or indirect) or nonmaternal (*12*). A maternal death was defined as a woman's death that was caused or contributed to by pregnancy and that occurred during pregnancy or within 1 year after the end of pregnancy (*4,13*). A direct maternal death was defined as a death resulting from complications of pregnancy, labor, or delivery or their management. An indirect maternal death was defined as a death in which pregnancy exacerbated a preexisting health problem. A nonmaternal death was defined as a death that occurred during pregnancy or within 1 year after the termination of pregnancy but was considered unrelated to pregnancy (e.g., due to injury, homicide, or suicide).

FIGURE 1. Areas reporting to the Maternal Mortality Collaborative, United States, 1980-1985



*San Jose only

The subjects' ages were grouped into 5-year periods for analysis. Race was classified as white or as black and other. Hispanics were classified as whites.

Maternal mortality ratios were calculated by using data on live births from NCHS (*14-19*). Instead of the standard term, "maternal mortality rate," the term "maternal mortality ratio" was used, which more accurately describes the statistic of maternal deaths per 100,000 live births. Maternal mortality ratio is a more accurate term because the numerator includes the number of deaths, regardless of pregnancy outcome (e.g., live birth, stillbirth, ectopic pregnancy), while the denominator includes only the number of live births. Numerators were matched with appropriate denominators by reporting area and year of occurrence.

RESULTS

Of the 714 deaths reported to the Collaborative for the years 1980-1985, 601 were classified as maternal and 111 as nonmaternal. In two instances, information regarding the cause of death was insufficient for classification. The maternal mortality ratio was 14.1 deaths per 100,000 live births. Complete statewide reporting was available for 18 of the 19 areas; in California, data were reported from only one city. For those areas of complete reporting, the Collaborative data, based on multiple sources, reflected 39% more maternal (direct and indirect) deaths for the 6-year period than did the NCHS data, based on death certificates alone (*6-11*).

Most of the decedents were married, were born in the United States, and had a live birth as the outcome of pregnancy (Table 1). The maternal mortality ratio increased with the age of the mother. Overall, women of black and other races had higher maternal mortality ratios than white women (Figure 2). For each age group, women of black and other races had a greater risk of dying than did white women; women of black and other races who were 30 years of age and older had the highest risk (12.6 times higher than that of white women 15-19 years old) (Table 2).

The leading causes of maternal deaths were embolism, indirect causes, hypertension in pregnancy, sequelae from ectopic pregnancy, hemorrhage, stroke, and anesthesia-related complications (Figure 3). The indirect maternal deaths resulted mostly from nonobstetric infections, cardiovascular disease, drug abuse, anemia, and diabetes. The leading causes of death differed by race (Table 3). Women of black and other races were more likely than white women to die from each cause, especially from complications related to ectopic pregnancy, anesthesia, and abortion.

The largest percentage of the deaths occurred during the first 24 hours after the termination of pregnancy (Figure 4). However, 50 (11%) of the deaths occurred more than 42 days after the pregnancy ended (Table 4). The timing of specific obstetric causes of death was unevenly distributed. Most of the deaths that occurred more than 42 days after the pregnancy ended resulted from indirect causes, cardiomyopathy, or embolism.

Of the 111 nonmaternal deaths, 90 (82%) were attributed to unintentional or intentional injuries. A majority (52%) of the injuries were unintentional, mostly due to automobile collisions. The other injuries were intentional, due to homicide or suicide (Figure 5).

DISCUSSION

The results from this study were similar to those of previous studies with regard to the increased risk of maternal death for older women and women of black and other

races (*4,6-11,20,21*). Separate analyses by age and race showed that for all age groups, black women had higher ratios of maternal death than white women. In particular, black women who were 30 years of age and older had a much higher risk of dying (12.6 times) than younger white women.

The Maternal Mortality Collaborative identified embolism, indirect causes, hypertensive disease of pregnancy, ectopic pregnancy complications, and stroke as the

TABLE 1. Selected characteristics of maternal deaths, 19 reporting areas, United States, 1980-1985

Characteristics	No.	Percent
All deaths	601	100.0
Age group (years)		
<15	0	0.0
15-19	62	10.3
20-24	144	24.0
25-29	152	25.3
30-34	151	25.2
35-39	66	11.0
40+	25	4.2
Unknown	1	
Race		
White	311	52.7
Black & other	279	47.3
Unknown	11	
Marital status		
Married	367	63.5
Not married	211	36.5
Unknown	23	
Birthplace		
United States	414	78.3
Foreign born	115	21.7
Unknown	72	
Outcome of pregnancy		
Full-term live birth	233	41.2
Premature live birth	109	19.3
Ectopic pregnancy	60	10.6
Stillbirth	57	10.1
Undelivered	74	13.1
Legal abortion	18	3.2
Spontaneous abortion	12	2.2
Self-induced abortion	1	<0.1
Gestational trophoblastic disease	1	<0.1
Unknown	36	

leading causes of maternal death. These findings also were similar to those of previous studies (*4*). Cause-specific mortality rates could not be calculated because appropriate denominators were not available for all causes of death. Instead, cause-specific mortality ratios were used to allow comparison with other published studies.

Maternal mortality ratios also differed by race for each cause of death. Previous studies have shown that women of black and other races have about three times higher mortality ratios related to complications of induced abortion and ectopic pregnancy than do white women (*22,23*).

Since 11% of maternal deaths occurred between 42 days and 1 year after the pregnancy ended, definitions of maternal death that limit the period between termination of pregnancy and death to 42 days may result in the incorrect classification of deaths that are caused by pregnancy or pregnancy complications.

Counts of maternal deaths and maternal mortality rates based on vital statistics are published annually by NCHS. The most recently published statistics are for 1986 (*24*). The NCHS counts are based entirely on the physician's certification of the cause of death on the death certificate, which NCHS processes and tabulates according to the

FIGURE 2. Maternal mortality ratios, by age group and race, 19 reporting areas, United States, 1980-1985

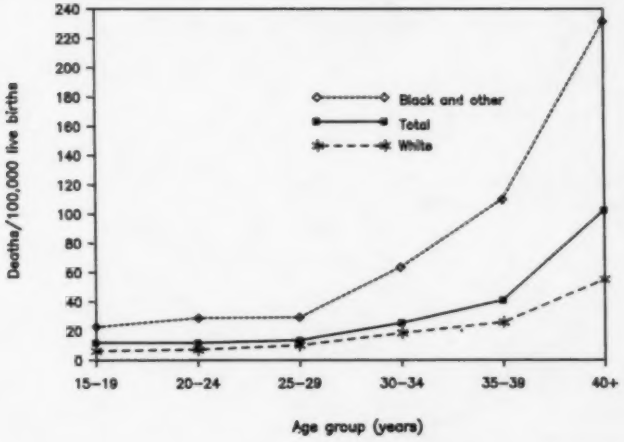


TABLE 2. Relative risk of maternal death, by age group and race, 19 reporting areas, United States, 1980-1985

	Age group (years)		
Race	15-19	20-29	30+
White	1.0 (referent)	1.3	3.2
Black and other	3.4	4.5	12.6

classification system, definitions, and rules for selecting underlying cause of death specified by the World Health Organization (WHO) in the ICD-9. According to WHO, "A maternal death is defined as the death of a woman while pregnant or within 42 days

FIGURE 3. Distribution of 601 maternal deaths, by cause, 19 reporting areas, United States, 1980-1985

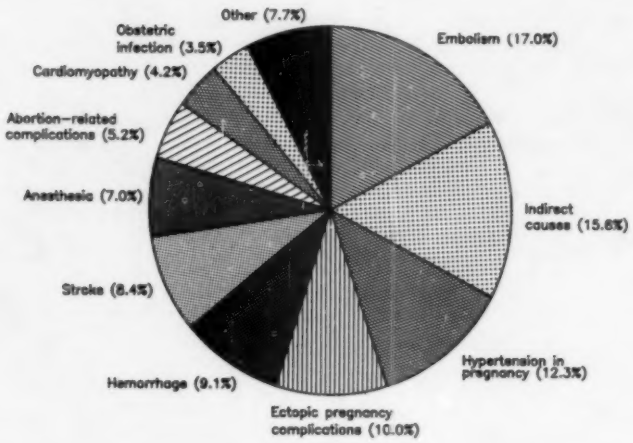


TABLE 3. Maternal mortality ratios* and relative risks, by race and cause of death, 19 reporting areas, United States, 1980-1985

Cause of death	Ratio*, by race		
	White	Black and other	Relative risk
Ectopic pregnancy complications	0.7	3.5	5.3
Anesthesia complications	0.6	2.3	4.2
Abortion (all types) complications	0.4	1.8	4.1
Cardiomyopathy	0.4	1.2	3.2
Hypertensive disease	1.1	3.4	3.0
Embolism	1.7	4.4	2.6
Indirect†	1.6	4.1	2.5
Obstetric infection	0.4	0.8	1.8
Stroke	1.3	1.8	1.4
Hemorrhage	1.1	1.5	1.3
Other	0.7	1.9	2.7
All causes	**10.0**	**26.7**	**2.7**

*Cause-specific maternal mortality ratio per 100,000 live births for each racial group.
†Indirect maternal deaths, coded as ICD-9 647-648.

of termination of pregnancy, irrespective of the duration and site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management but not from accidental or incidental causes."

Death from injury accounted for most of the nonmaternal deaths. In the United States, intentional and unintentional injuries are a leading cause of death for reproductive-age women (*25,26*). In one state, studies of maternal mortality revealed

FIGURE 4. Maternal deaths, by number of days from time of delivery, 19 reporting areas, United States, 1980-1985 (N=601)

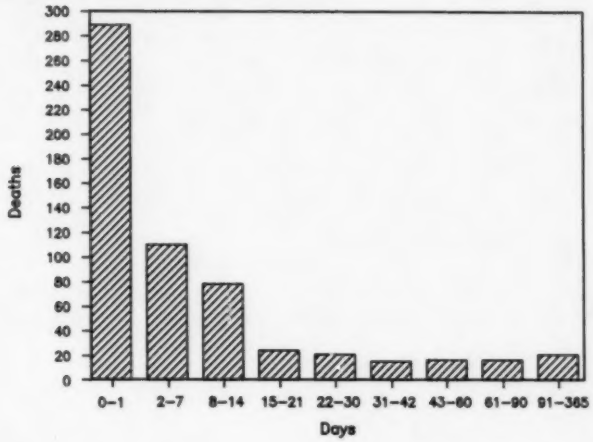


TABLE 4. Number and percentage of maternal deaths that occurred more than 42 days after pregnancy ended, 19 reporting areas, United States, 1980-1985

Cause of death	No.	Percent
Embolism	7	14.0
Hypertensive disease	1	2.0
Ectopic pregnancy complications	2	4.0
Hemorrhage	2	4.0
Stroke	2	4.0
Anesthesia complications	4	8.0
Abortion (all types) complications	3	6.0
Cardiomyopathy	11	22.0
Obstetric infection	1	2.0
Gestational trophoblastic disease	2	4.0
Other direct causes	3	6.0
Indirect causes	12	24.0
TOTAL	**50**	**100.0**

that injury was the leading cause of nonmaternal deaths for 1982-1985 (*27*). Some nonmaternal injuries probably are missed because these deaths are not routinely identified and analyzed by maternal mortality committees (*28*).

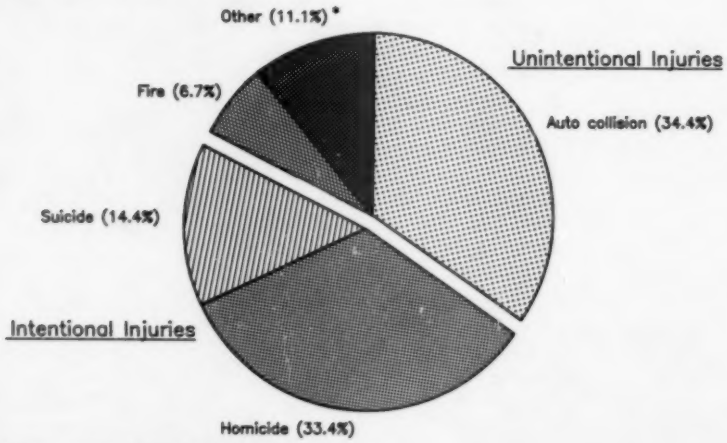
The results of this study suggest that more complete information on maternal deaths may be obtained from a voluntary reporting system that uses intensive case detection and reporting by multiple sources than from national vital statistics alone. The number of maternal deaths identified in this study was 39% higher than that published by NCHS for the same reporting areas and time period. This discrepancy was due, in part, to the different definitions of maternal mortality; NCHS counted only those deaths that occurred within 42 days after termination of pregnancy, and the Collaborative counted deaths that occurred up to 1 year after termination. However, even after an allowance was made for the difference in definitions, the Maternal Mortality Collaborative still reported 29.5% more maternal deaths than did NCHS.

Even though data from the Collaborative are more representative than those for any single state or region, they cannot be generalized to the total United States population.

Although the definition for maternal death used by the Collaborative was broader than that used for national vital statistics, an understatement of maternal deaths still could have occurred. Misclassification of maternal deaths has been well documented in a number of nationwide and statewide studies (*3,29-30*). In addition, information submitted for many of the cases was derived only from death certificates, which provided scant information about causes of death, risk factors, and obstetrical history; therefore, only limited conclusions about the risk of maternal death could be drawn.

At present, vital statistics collected by NCHS are the only source of information about U.S. ratios of maternal mortality. Until January 1988, no systematic, national

FIGURE 5. Distribution of deaths due to injury, by cause, 19 reporting areas, United States, 1980-1985 (N=90)



*Includes unspecified injuries, falls, and drownings.

system existed for studying the epidemiology of maternal mortality. Some states collect and publish state ratios of maternal mortality; however, both the national and the state vital statistics systems have several limitations.

First, information reported from vital statistics alone is limited. Death certificates alone may not provide adequate information on the sequence of events that led to death. Ultimately a single code is assigned to classify the underlying cause of death. Often, however, several factors may contribute to a death; therefore, the death cannot be adequately described with a unidimensional code (*31*). In this study, although we used a single code for classifying each of the deaths, we also assigned multiple causes of death if appropriate. Almost half of the cases (48%) had multiple cause of death codes used to describe the series of events leading to death.

Second, no standard, scientifically valid definitions of maternal death are used by all researchers and clinicians. The differences in the period between termination of pregnancy and death used in defining maternal mortality is one example of a definitional difference that can cause variations in case detection and reporting. NCHS uses 42 days in its definition (*32*); the American Medical Association, 90 days (*33*); the American College of Obstetricans and Gynecologists, 42 days (*34*); and the Maternal Mortality Collaborative, 1 year.

Finally, the coding system used by national vital statistics and states to describe maternal deaths includes a combination of outcomes of pregnancy (e.g. ectopic pregnancy, abortion), immediate causes of death (e.g., hemorrhage), and underlying obstetrical conditions that contribute to death (e.g., obstructed labor). This system of classification precludes a determination of the real causes of maternal death. To develop strategies to prevent maternal deaths, public health personnel need to know the immediate cause of death as well as the underlying conditions that led to death.

Because of the success of this voluntary reporting system, the Division of Reproductive Health, CCDPHP, initiated National Pregnancy Mortality Surveillance in January 1988 (*35*). This system was designed to overcome some of the limitations inherent in the present systems. In the new system, multiple sources of reporting and of information are used to identify and investigate all reported pregnancy-associated deaths in the United States. Sources included vital records; reports from maternal mortality committees, practicing physicians, and private citizens; medical examiners' reports; scientific literature; and special studies or surveys. This surveillance system also introduces a new method for assigning causes of death to each case upon complete investigation. Death certificates of pregnant women are matched with birth certificates and fetal death certificates to provide a basis for analysis. In addition, autopsy reports, coroners' reports, and medical records are used to determine the cause of death and the factors that placed women at increased risk of maternal death. Through this new system, the outcomes of pregnancy will be analyzed separately from the immediate causes of death. For example, if a woman died from complications of ectopic pregnancy because of a ruptured fallopian tube that led to hemorrhage, the immediate cause of death (hemorrhage) would be analyzed as well as the pregnancy outcome (ectopic pregnancy).

The success of this new system will depend on data reported from various sources, such as state health departments, maternal mortality committees, and individual practitioners. More detailed information about this system may be obtained by calling the Pregnancy Epidemiology Branch at (404) 639-3131 or the Research and Statistics Branch at (404) 639-3392, or by writing to Pregnancy Mortality Surveillance, Division

of Reproductive Health, Center for Chronic Disease Prevention and Health Promotion, Mailstop CO6, Centers for Disease Control, Atlanta, Georgia 30333.

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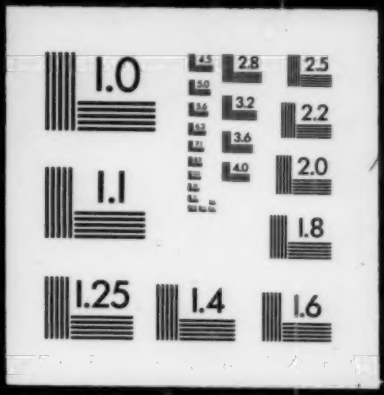
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